

CONCLUSIONS. 1. Evidence has been presented that in a certain proportion of diabetics beyond middle age the metabolism and general symptoms may be improved and the sugar removed from the urine, at least temporarily, through the administration of an infusion of lean meat acidulated with hydrochloric acid.

2. In a single case of youthful diabetes (Case VII), though neither beef infusion nor pancreatic infusion was alone efficacious, when the one followed the other, or a mixture of the two after an interval of some hours, the urine became sugar-free. After the disease had persisted for some months this happy result could no longer be obtained. Nevertheless, the treatment, especially with the beef infusion, seemed to improve the subjective condition of the patient.

3. No good results attended the use of the commercial pancreatic powder employed.

PASSING LEAKAGE OF THE PULMONARY VALVE.*

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INCOMPETENCE of the pulmonary valve due to organic disease is a very rare condition. Only 24 cases were noted at Guy's Hospital out of 16,000 postmortem examinations during a period of thirty-two years,¹ and 3 among 24,000 cases admitted to the medical wards of Johns Hopkins Hospital.² But leakage may occur much more commonly here when the flaps of the valve are normal, but the ring to which they are attached is so dilated that the normal flaps are no longer able to close the enlarged orifice. It is with this latter condition, the so-called relative incompetence, that we are here concerned.

Relative incompetence is a condition not easy to demonstrate post mortem, and many cases, no doubt, occur during life, and have been carefully described, in which the valve was found at the autopsy to be competent. R. B. Preble³ and others have published cases in which post mortem the pulmonary valve was incompetent and would not hold water, and yet the flaps were normal in every way; but it is easy to understand that, as long as any elasticity remains in the structures about the pulmonary orifice, this ring will tend to return to its normal size as soon as the pressure stretching it is removed. Thus, a valve which has leaked freely during life may post mortem appear to be normal, and may even hold water,

* Read by title before the Association of American Physicians, May 10, 1911.

which, after all, is at almost no pressure. In the writer's opinion such temporary leakages are far from uncommon, and he notes with pleasure a recent statement of Dr. Thayer to the same effect.⁴

Relative incompetence must be due either to (a) an increased blood pressure within the pulmonary artery and right ventricle, or to (b) a want of resistance to stretching of the tissues about the orifice. The question of exactly what tissues may be here involved need not be gone into, but Hugh Stewart two years ago demonstrated before the Association of American Physicians the importance of the muscular ring below the aortic orifice in preserving the integrity of that orifice, and, according to G. A. Gibson,⁵ Arthur Keith has shown that a loss of tone in the muscular fibers surrounding the mouth of the pulmonary vessel allows of stretching of the fibrous tissues.

Most cases of relative incompetence of the pulmonary valve that have been described have occurred as a sequel to mitral disease, especially mitral stenosis (Graham Steel) and fibrous disease of the lungs. It has been shown by Foxwell, Stacey Wilson, and others how easily the pulmonary artery dilates under internal pressure as compared to the aorta—six times as easily, after making all allowance for the different strains under which they work—and a number of workers have further endeavored to find the amount of pressure required to dilate the pulmonary orifice so as to produce leakage there.

G. A. Gibson found that a pressure of $14\frac{1}{2}$ inches of water was sufficient to cause a strong jet of water to escape through the pulmonary orifice in a sheep's heart; and in the same way a healthy human heart leaked freely at a pressure of 13 inches of water, and slightly with the pressure down to 8 inches.

We have tried a number of hearts in the same way, and on the whole with about the same results.

A bullock's pulmonary valve leaked freely at a water pressure of $6\frac{1}{2}$ feet, but long before this pressure was attained the pulmonary artery was ballooned in a marked fashion. When a string was loosely put round the orifice before the pressure was raised, no leakage occurred up to nine feet.

Seven human hearts were similarly tested, and they leaked freely under pressures varying from 11 to 18 inches, and often below that amount to a slight degree.

When the pressure is gradually raised in the pulmonary artery, the vessel very easily dilates in a balloon-like manner, the sinuses of Valsalva standing out with special prominence. Then after a certain pressure is reached a jet of water escapes from a small opening which appears at the apices of the flaps of the valve. If, however, one encircles the orifice with the fingers or with a previously adjusted ligature, the pressure may be raised to several feet without any incompetence of the valve occurring. This would suggest that

the leakage is due to dilatation of the ring around the valve, and not to any distortion due to the dilating of the pulmonary artery. Under Professor Brodie's suggestion the latter experiments were done without any previous alteration of the relations of the pulmonary artery to the aorta or heart, and finally with the heart still *in situ* in the cadaver and with the pericardium unopened. These precautions did not make any appreciable difference in the results.

In none of the experiments could the aorta be made to dilate to any extent, even under a pressure of 20 feet of water, much less could the aortic valve be made to leak. There was one exception to this in the case of a heart from a boy, aged two years, who had died of whooping cough, with complicating pneumonia. Here the aorta leaked at 8 feet, and before the leakage occurred the aorta did not dilate much. The endocardium about the orifice was greatly congested, as was also the mitral valve.

As the pulmonary artery dilates so easily even under a moderate pressure (and in these experiments it is shown that after the dilatation reaches a certain point the orifice shares in it so that leakage occurs), one would expect that such dilatation and even leakage would commonly occur during life, always with the proviso that the tissues dilate during life with anything like the pressure that they do in the cadaver. The mean blood pressure in the pulmonary artery is 17.6 mm. of mercury in the cat, 12.07 mm. in the rabbit, 29.6 mm. in the dog, according to Bentner,⁵ and Gibson says that "pressure in the pulmonary artery in man at the height of a vigorous systole of the right ventricle may be well above that which first causes an insufficiency." No one believes that the normal pressure ever makes a normal pulmonary valve leak, but if the pressure be raised or the tissues be relaxed, then dilatation of the artery may and can often be demonstrated to have occurred; and if the orifice shares in this stretching, leakage may be produced even to a sufficient extent to cause a murmur, such a yielding of the orifice being no doubt largely due, as Stewart and Keith have urged, to a want of tone in the muscular ring just below the orifice.

The following three cases are ones in which an evident mitral lesion so raised the pulmonary artery blood pressure that that vessel and along with it the pulmonary orifice dilated and a murmur occurred, which came and went in two of them over a period of years.

CASE I.—V. A., girl, aged thirteen years. Admitted to the Victoria Hospital for Sick Children last August, suffering from pericarditis and double mitral disease, with a marked accentuation of the pulmonary second sound.

October 18 the following note was made: Pulmonary second sound still much accentuated, and is now accompanied by a distinct short murmur. This murmur is localized to an area about the size of a penny over the pulmonary area and a little below it. It is unaffected by holding the breath.

October 26. Whole precordium heaves with each pulsation of the heart. On auscultation a double mitral murmur is heard; also a pulmonary diastolic.

November 6. Mitral murmurs only present. Pulmonary second much accentuated.

November 24. Diffuse heaving pulsation of the whole precordium. In the second left interspace two pulsations are seen, of which the outer is the earlier. (The outer is probably the left auricle and the inner the dilated pulmonary artery.) Double mitral murmur present, but no diastolic murmur, with the pulmonary second. This sound is much accentuated.

CASE II.—A. P., girl, aged fifteen years. When seen on February 19, 1908, she was suffering from fever and pains in various joints. Within a few days the heart became markedly irregular and a mitral systolic murmur developed. This murmur became gradually more marked, and was present in all postures of the body. The pulmonary second was accentuated.

Two months later chorea developed. The mitral murmur was still present, and had become almost musical in character.

In June (two months later) it was noted that chorea was still evident, and the patient has developed flat foot. Mitral murmur still present, and the pulmonary second sound is markedly accentuated. A slight diastolic murmur is audible down the sternum. It is not heard in the neck. The heart is still irregular, giving an occasional quick beat. Apex is in the fifth intercostal space, just outside of the nipple line. No thrills. When patient sits up the second sound at the base becomes double and the diastolic murmur is more evident.

July 19. White blood cells, 5000. Heart again somewhat irregular. Diastolic murmur about the third left costal cartilage very evident.

October 16. Mitral systolic well marked. Pulmonary second sound much accentuated, and no murmur accompanies it in any posture. Cardiac deep dullness reaches from one-half inch beyond right of sternum to the nipple line. Apex in nipple line in the fifth space.

April 15, 1909. Feels well, and choreic movements are practically gone. Mitral systolic well marked, as is also the pulmonary second sound, which, however, is pure.

July 20, 1910. Seems quite well, but heart rather wide upon percussion, and mitral systolic murmur evident, and the pulmonary second sound is plus and accompanied by a distinct murmur. The pulse is not water-hammer in type, nor is there any bounding of the vessels in the neck.

CASE III.—Female, aged thirty years. Came on April 4, 1904, complaining of cough and shortness of breath, which had been frequent during the last few years. The note then made was that

some bronchitis is found. Cardiac apex in the fifth space in nipple line. Here, first sound is flapping and a slight presystolic murmur is audible. A diastolic bruit is heard over lower part of sternum, but no capillary or water-hammer pulse present, nor are the vessels in neck bounding.

February 19, 1905. Presystolic murmur not heard, but a slight diastolic one audible, best marked over lower part of sternum. First sound sharp at apex.

November 19, 1906. Has had one of her frequent attacks of bronchitis. Careful examination of the heart reveals a marked presystolic murmur at the apex, with a flapping first sound. Absolutely no other murmur is audible, and the second sound is pure everywhere.

February 19, 1907. Bronchitis again. Diastolic murmur again, audible about the lower part of the sternum. Apex is in nipple line, in fifth space. No murmur heard in the neck, and here the second sound is well heard and is free from impurity.

May 15. First sound flapping at apex. Second sound is pure everywhere.

These three cases are all examples of the so-called Graham Steell murmur, in that there was present well-marked mitral disease, which presumably so raised the blood pressure in the pulmonary artery as to cause from time to time leakage at the pulmonary valve. In the following cases, however, no such cause seemed to exist, and probably the leakage was more due to relaxed tissues about the pulmonary orifice than to any marked increase in the blood pressure of the pulmonary artery.

CASE IV.—Mrs. C., aged thirty years. Was first seen in March, 1904, for a slight attack of appendicitis. It was then noted that she had an aortic diastolic, with well-marked second sound. Pulse was regular and not of the water-hammer type.

Two years later, in November, 1906, aortic diastolic murmur not heard. Heart sounds are all quite clear and pure.

April 5, 1907. Aortic diastolic murmur now quite evident.

Dr. Finlay, of Montreal, had previously had this patient under his care, and on my writing to him about her, he replied as follows: "I remember Mrs. C.'s case quite well. She had a soft diastolic murmur, localized about the pulmonary area, but without the compensatory enlargement or the vascular phenomena of aortic incompetence. I always felt a little uncertain as to the significance of the physical signs. I can recall another case of a diastolic aortic murmur in which at several subsequent examinations it was inaudible."

March 17. The diastolic murmur is quite evident. The heart does not appear to be enlarged.

November 13, 1910. The pulse is regular and not water-hammer in type. No throbbing in the neck. Cardiac apex is in fifth space

one inch outside of the nipple line. The sounds are clear at the apex, but down the left border of the sternum there is a slight diastolic murmur. The pulmonary second sound is accenutated. There is no murmur in the neck. The liver is of normal size.

In this case, as the notes show, both Dr. Finlay and myself presumed that the murmur was of aortic origin, but I now believe that it was an example of occasional leakage at the pulmonary orifice.

CASE V.—G. W. H., aged fifty-five years; farmer. Seen last November, with Dr. Brown, of Elmwood. Suffered from pernicious anemia of at least two years' duration. His chief complaints were of weakness and dyspnea. This note was made: Pulse regular and not of water-hammer type. Cardiac apex in fifth intercostal space half an inch outside of nipple line. No epigastric or venous pulse. Right border of cardiac dulness does not reach beyond sternum. At the apex there is a slight systolic murmur, and the second sound is clear. In the aortic area the sounds are both clear, and the second is not accentuated. In the pulmonary area the second sound is much accentuated, and is accompanied by a well-marked diastolic murmur. This is best heard about the inner part of the second left intercostal space, but can be heard for about an inch below this point. It persists through all phases of respiration, but is loudest at the end of expiration, and when the breath is then held. A well-marked pulmonary systolic murmur is also present.

Of course, in this case some old organic lesion of the pulmonary valve may have been present, but this is unlikely both on account of the rarity of such and also from the fact that until two years before the patient had been in rugged health.

DIAGNOSIS. The chief classical sign of pulmonary regurgitation is a diastolic murmur best heard on the left side of the sternum, chiefly at the end of expiration, and not audible in the vessels of the neck nor at the cardiac apex. Along with this murmur there are signs of enlargement of the right heart with displacement of the apex toward the left. There is an absence of the water-hammer pulse and of a capillary pulse. The pulmonary second sound may be weak or absent, all depending upon whether or not the diseased flaps are able to close.

In the cases, however, where the leakage is due not to organic disease, but to relative insufficiency of the valve, the first thing usually noted is the accentuation of the pulmonary second sound, which loud sound is from time to time accompanied by a murmur of the above distribution. In all the cases so far noted by the writer the murmur has not been constant, but no doubt it often is persistent, especially when due to some permanent obstructive condition farther on in the pulmonary circulation, such as mitral stenosis or pulmonary fibrosis.

PROGNOSIS. The importance of relative insufficiency of the pulmonary valve, of course, depends upon the fundamental cause of

the leakage. Where this is mitral stenosis—probably the most common permanent antecedent of the condition—it points to a considerable obstruction at that orifice, but even then the cases may do well for years, as in Cases II and III of our series, and after several years of known pulmonary leakage seem little the worse.

Where the leakage is due rather to a relaxed condition of the tissues about the pulmonary orifice the prognosis depends entirely upon the cause of this relaxed state. Dr. Thayer⁴ mentions the condition as occurring in exophthalmic goitre, and as this disease lessens, one would expect the pulmonary leakage to stop.

A pulmonary systolic murmur, the commonest of all functional bruits, probably depends upon a dilatation of the pulmonary artery, and in most cases tends to disappear. When, however, the dilatation involves the pulmonary orifice so that a diastolic leakage occurs the condition is of more import, although even yet it may completely clear up.

From the dilatable character of the pulmonary orifice, and on the other hand the resistant nature of the aortic one, it seems probable that most of the diastolic murmurs which come and go—and which are usually reported to be aortic in origin—are due to passing leakage at the pulmonary valve.

REFERENCES.

1. Pitt, G. Newton. Allbutt and Rolleston's System of Medicine, vi, 310.
2. Herschfelder, A. H. Diseases of the Heart and Aorta, p. 390.
3. Preble, R. B. Jour. Amer. Med. Assoc., 1897, p. 1012.
4. Thayer, W. S. Trans. Assoc. Amer. Phys., 1910, p. 75.
5. Gibson, G. A. Arterial Pressure, Edinburgh Med. Jour., March, 1911.
6. Osler and McCrae. System of Medicine.

FURTHER CLINICAL STUDIES IN THE AUSCULTATORY METHOD OF DETERMINING BLOOD PRESSURE.¹

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In a previous paper² we described in full the auscultatory method of determining blood pressure, giving details of technique, with explanation of sounds heard, and calling attention to the importance

¹ Read before the College of Physicians, May 3, 1911.

² Univ. Penna. Med. Bulletin, November, 1910.